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Environmental Carbon Monoxide from Cigarette Smoking

L'Oxyde de Carbone Provenant de la Fumée de Cigarette, dans le Milieu Ambiant

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SUMMARY

The current drive for bans on smoking in public places and in work places is based on the assertion that tobacco smoke in the atmosphere constitutes a health hazard to exposed non-smokers. It is said that the hazard arises mainly from carbon monoxide generated during the burning of a cigarette.
Cigarette smoking is an insignificant source of carbon monoxide in the atmosphere as compared to other natural and man-made sources. Even in tightly closed spaces with a large percentage of smokers, only rarely is it possible to build-up concentrations which would exceed the established threshold limiting values for extended exposures. Examples including meeting rooms, houses, buses, and aircraft will be discussed. The resulting carboxyhemoglobin levels in nonsmokers will be considered. It is concluded that the carbon monoxide from environmental tobacco smoke is well below the amount needed to produce the maximum allowable limit of 4 % carboxyhemoglobin in the blood as suggested by the World Health Organization.

RÉSUMÉ

La campagne actuelle pour l'interdiction de fumer dans les lieux publics et de travail se base sur l'assertion que la fumée de tabac dans l'atmosphère constitue un danger pour la santé des non-fumeurs qui y sont exposés. On dit que le danger vient de l'oxyde de carbone engendré par la cigarette allumée.
La fumée de cigarette est une source insignifiante d'oxyde de carbone dans l'atmosphère comparée aux autres sources naturelles ou industrielles. Même dans des lieux clos, avec un pourcentage élevé de fumeurs, il n'est que rarement possible d'élever la concentration de CO à des niveaux dépassant ceux qui ont été établis comme valeurs-seuils pour l'exposition extensive. Parmi les exemples évoqués, on trouvera les salles de réunion, les maisons d'habitation, les autobus, et les avions. Les taux de carboxy-hémoglobine chez les non-fumeurs seront considérés. On estime, en conclusion, que l'oxyde de carbone dans le milieu, qui procède de la fumée de tabac, est bien en dessous de la quantité nécessaire pour produire les 4 %, considérés comme maximum par l'O.M.S., de carboxy-hémoglobine dans le sang.

INTRODUCTION

During the past decade the anticigarette movement has undergone a basic change in direction. Instead of attacking the smoker, it has now become the self-appointed protector of the nonsmoker. In former times, even centuries ago, those opposed to smoking traditionally argued that smoking was harmful to the smoker. This viewpoint even had Judeo-Christian foundations and took on religious overtones. It was argued that in smoking (as well as

in drinking, taking drugs, eating unclean foods, or engaging in nonprocreative sex) your body, « the Temple of the living God », (1) was being defiled. Many elements of our modern free society have rejected this attitude. They have come to accept the notion that the individual is free to do as he pleases so long as he does not harm his neighbor.

Now, the anticigarette movement has cleverly seized upon this new approach to justify restricting the smoker. Cigarette smoke in the environment, the anticigarette activist says, is polluting the air so as to harm the nonsmoker. Smoking must be banned in all places where there is any possibility that smoke might be inhaled by innocent nonsmokers. Recently, there has even been proposed to the USA Federal Occupational Safety and Health Administration (2) that smoking should be banned in all *work places*. A *work place* is defined as a location where two or more persons are working. The basic rationale for thus restricting the freedom of the smoker is that in smoking he may be causing harm to someone else. The smoker is made to feel guilty in the presence of nonsmokers. He is even said to be mentally ill (or perhaps deranged) because of his insistence on hurting himself and others. The nonsmoker is being encouraged to bring pressure on the smoker to refrain from smoking.

This new move to protect the nonsmoker has brought forth a rash of studies to demonstrate how it might be that the nonsmoker is being harmed. By far the majority of these investigations is focused on carbon monoxide (CO) generated by the burning cigarette. In this paper I shall briefly examine those factors relating to the effects of environmental CO from cigarette smoking.

SOURCES OF CARBON MONOXIDE

It has been estimated that the earth's atmosphere at any given moment contains approximately 530 million metric tons of CO (3, 4) (Table 1). Natural and manmade sources produce annually ten times this amount. Because of the continual consumption of CO in a number of oxidation reactions in the atmosphere and stratosphere, and in microbial actions in the soil, the half life of atmospheric CO is only one-tenth of a year.

Note that cigarette smoking worldwide is estimated to produce only about 0.6 million tons of CO per year, an infinitesimal amount as compared to other sources. This estimate is based on an average figure of 0.100 gram per cigarette for both mainstream and sidestream smokes. Clearly if we are to be concerned about CO from smoking, it must be from a local concentration point of view rather than from the total amount available in the atmosphere. For this reason, attention is usually focused on smoking in enclosed spaces such as rooms, buses, automobiles, airplanes, arenas, etc.

TABLE 1

Annual production and losses of atmospheric carbon monixide (3, 4)

Production	Estimated production or loss in millions of tons
Atmospheric oxidation of methane	5,000.
Combustion of fossil fuels	270.
Escape from oceanic solution	150.
Tobacco smoking	0.6
Losses	
Atmospheric oxidation to CO_2	4,500.
Stratospheric oxidation	500.
Soil microorganisms	500.
Equilibrium content	530.

CONCENTRATIONS OF CO IN CLOSED SPACES

Carbon monoxide concentrations in nonurban outdoor atmospheres are usually less than 2 ppm. In highly populated cities average concentrations may range as high as 10 ppm (5), depending on proximity to sources (highways or streets, factories, etc.) and air circulation (or stagnation). In general, outdoor concentrations are less than the ambient air quality standard of 9 ppm or the USA Environmental Protection Agency standard limit of 8 ppm for 24-hour exposure (6).

In enclosed spaces such as homes, offices, elevators, workshops, autos, buses, aircraft, etc., maximum equilibrium concentrations of CO buildup from smoking depend on rates of cigarette smoking, volume of space, ventilation rates, and CO losses(7). A large number of combinations have been investigated and measured(8-11). Unfortunately, many of the tests were carried out under extreme conditions that

are completely unrealistic in terms of human exposures.

Some recent results are listed in Table 2. It is immediately evident from these data that under realistic conditions, CO concentrations from smoking are only one-fifth to one-tenth of those reported in sealed climatic chambers or unventilated rooms. Both RUSSELL (10) and HARKE (11, 12) found that in these closed, unventilated, smoke-filled rooms people experience annoying eye and nose irritations long before the CO concentration in the room reaches 40 ppm.

The American Conference of Governmental Industrial Hygienists (13) has established a Threshold Limiting Value for CO of 50 ppm as the maximum for daily 8-hour exposure. JONES and FAGAN (7) have shown that in a closed room or house it is almost impossible to attain this concentration of CO by smoking alone. The reason for this is that unless the space is tightly sealed, « normal » leakage through cracks around doors and windows permits substantial circulation of air into and out of the room (14), usually sufficient to change the total volume of air at least once every hour. There is, thus, an upper limit to the possible buildup of carbon monoxide concentration from smoking in the real-life situation. Many experimenters who have studied the smoke-filled room have found this out, often to their surprise and dismay.

It is also of interest to note in this connection that the modern jet passenger aircraft is probably the most effectively ventilated enclosed space occupied by humans, since a complete change of air occurs every three minutes. Even if all the 165 passengers in a full Boeing 707 aircraft were smoking at the rate of four cigarettes per hour, the ambient carbon monoxide concentration would only increase by 12 ppm. The actual levels are much lower than this value (15).

In the arguments used to justify the imposition of laws restricting the rights of smokers to smoke only in restricted places, only those investigators who used extreme and unrealistic test conditions are most often cited. Exaggerated claims are made that CO from smoking greatly harms nearby nonsmokers. No one suggests that the best control for carbon monoxide from smoking in any indoor space is simply to provide reasonable ventilation, rather than restrict the freedom of the smoker (16).

INHALED CO AND CARBOXYHEMOGLOBIN

The main effect (if not the only effect) of

TABLE 2

Maximum CO concentratings recently reported in enclosed spaces

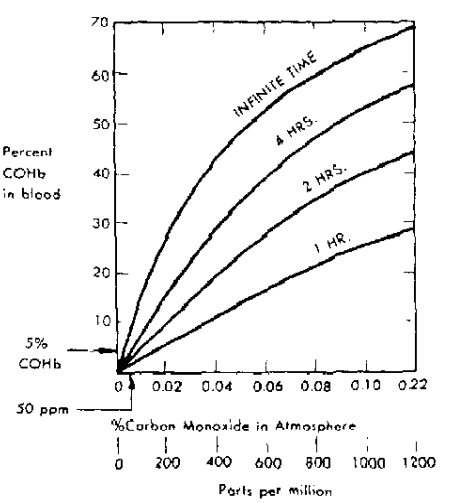
Situation	Estimated smoke generation rate Cigs/m^3/hr.	Ventilation Air changes/hr.	Maximum CO ppm	Reference
With little or no ventilation				
Room 58 m^3	2.6	None (?)	50	(46)
Room 43 m^3	1.4	None (?)	38	(10)
Small autos on Hamburg streets	~3.0	Vents closed	24.3	(47)
Stationary auto	~3.0	Vents closed	110.4	(48)
Unventilated climate chamber 30 m^3	6.0	0.1	88 @ 10 min.	(49)
With normal ventilation				
Room 58 m^3	2.6	8.8	10	(46)
Party of 50 people in 170 m^3 room	0.26	7.0	7	(50)
Room 80 m^3	0.3	6.4	6	(51)
Small autos on Hamburg streets	~3.0	Vents open	12.0	(47)
Stationary auto in wind tunnel	~3	~60	5-6	(48)

inhaling CO is the formation of carboxyhemoglobin (COHb) in the blood (Figure 1). CO binds strongly to the hemoglobin preventing it from performing its intended function of transporting oxygen to the tissues and carbon dioxide to the lungs. The level of COHb (expressed as a percent of the hemoglobin so taken up) depends upon the concentration of CO in the lungs, the time of exposure, the rates of respiration and heart beat, exercise factors, etc. (17-21). Eventually under constant conditions an equilibrium concentration of COHb is established (22, 23). The formation of COHb is reversible so that at lower concentrations of CO, the COHb breaks down to release free hemoglobin again. Obviously, at high enough levels of COHb (50-70 %) the oxygen needed by various tissues is no longer supplied in adequate amounts and various adverse effects on body functions are observable. That such effects do not take place from inhaling cigarette smoke will be shown later.

Under these relationships between CO from smoke and COHb, the only important question is : How much COHb is formed in the exposed subjects? In fact this is the only complete statement one can make about CO exposure. Statements limited to transient concentrations of CO in smoke or in the atmosphere are inadequate to define what is taking place. Inferences from such marginal information about the effects of CO are usually based on unrealistic assumptions concerning the other conditions involved. For this reason, most of the information in the literature about CO concentrations from smoking is meaningless in terms of human experience.

Figure 1

Effects of high levels of carbon monoxide (56). Note that in the lower left hand corner are shown the values of 5 % COHb, the level found for the average smoker in an urban environment; and the atmospheric concentration value of 50 ppm, the Threshold Limiting Value established by the American Conference of Governmental Industrial Hygienists.



OBSERVED COHb CONCENTRATIONS

It is impossible in the scope of this paper to review all the studies which have been made on the COHb concentrations in smokers and in nonsmokers. Such investigations are invariably complicated by the fact that differing CO concentrations are found in various environments even in the absence of smoking. In fact, the human body itself generates CO *de novo* so that the blood always contains from 0.4 to 1 % COHb (9, 24).

Table 3 summarizes some of the recent findings.

The pattern from these data is very clear. Nonsmokers will generally have less than 2 % COHb while smokers will average about 6 %, ranging from 2 to 13 % depending upon the extent and manner of smoking and on other environmental factors. Only in extreme situations such as those used by Russell in his experiments will nonsmokers experience COHb

TABLE 3

Carboxyhemoglobin Levels in Smokers and Nonsmokers

Description	Mean (m) or Range (r) of % COHb	
	Nonsmokers	Smokers
UK pregnant women	m 1.1%	m 3.6%
Meat porters	m 1.6	m 5.1
Office workers (17)	m 1.3	m 6.2
London office workers (52)	m 1.12 r 0.1-2.7	m 5.5 r 2.2-13.0
29,000 USA blood donors (53)	m 1.39 r 0.4-6.9	m 5.57 r 0.8-11.9
3311 California longshoremen (54)	m 1.3	m 5.9
Munich population	m 2.36	m 7.38
Rural Bavarians (55)	m 1.03	m 6.06

levels in excess of 2 % from cigarette smoke-filled environments.

RUSSELL's (10) description of such an experiment is a classic :

"Twenty-one research and clerical colleagues volunteered to provide blood specimens before and after spending about 1 hour in a smoke-filled room. The conditions were deliberately made worse than would be likely to be encountered in natural social situations. The room was approximately 43 m^3 (15 x 12 x 8 ft.). Ventilators were switched off and all windows were closed. Before the volunteers entered the room it was « smoked up » by leaving thirty cigarettes (« Piccadilly » tipped) to burn in ashtrays. During the experiment the smokers smoked thirty-two cigarettes and two cigars ; a further eighteen cigarettes were left to burn in ashtrays. During the average time of 78 minutes spent in the room the volunteers remained seated in the same place. The exposure was very unpleasant, causing eyes to burn and water. For most volunteers it was worse than they could recall having tolerated on normal social occasions."

Imagine, if you will, this experimental situation. The room had only 18 square meters of floor space, occupied by 21 persons. In 78 minutes smoke was generated from 80 cigarettes and two cigars. None was allowed to escape through ventilation. After all this, the maximum CO concentration in the room was only 38 ppm. For the nonsmokers the average COHb had increased from 1.6 % to 2.6 %. One wonders what the room conditions would have been even without smoking.

The World Health Organization (25) has suggested a maximum of 4 % COHb as a level which is safe from all possible adverse effects under continuous exposure conditions. In order to reach this level, the non-smokers in Russell's experiment would have had to remain in the intolerably smoky room at least another 78 minutes.

HARKE (11) carried out similar experiments and made the following concluding statement :

"It does not appear possible that such smoke densities can be generated by smokers This experiment using smokers (rather than smoking machines) would only be reproducible when 50 persons were placed in the room (floor space 62 m^2), and each of them would smoke three cigarettes during a period of 30 minutes. The irritation....would be so great that certainly a large majority of the smokers would have to leave the room prior to the end of the smoking

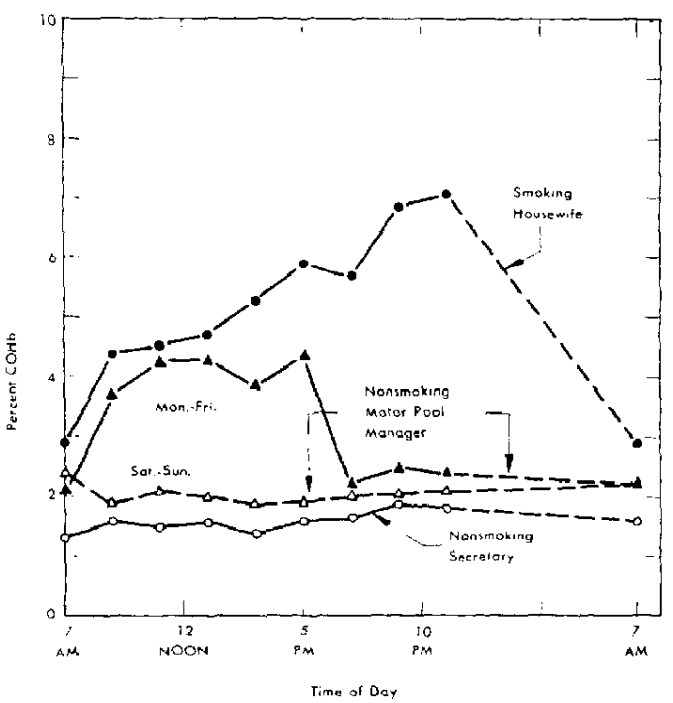


Figure 2

Temporal variations of COHb concentration (26). Note that the COHb level of the nonsmoking motor pool manager increases during the day when he is at work and quickly drops again at the end of the work day. The smoking housewife would have a daily average of about 5 % characteristic of smokers, the value shown in the lower left hand corner of figure 1.

period."

It should be emphasized that because of the dynamic interchange of CO between the inhaled air and the COHb in the blood, the level of COHb is constantly changing, depending on circumstances. McILVAINE and associates (26) have measured COHb values at two-hour intervals throughout the day for smokers and nonsmokers. Some results are shown on Figure 2. Obviously, the definition of an average condition of exposure to carbon monoxide presents some difficulties.

HUMAN RESPONSE TO INCREASED COHb

Many investigators have carried out performance and pathological tests aimed at showing that increased COHb in the blood impairs the well being of the persons exposed to CO in the environment (27-29). Performance tests are generally directed to demonstrating the impairment of oxygen sensitive organs such as the eye, the brain, and the central nervous system. These researches indicate that no discernible effects occur in normal healthy adults below 10 % COHb. Some effects may be observed at relatively high and unusual COHb levels (15 % and over).

Recently, a number of studies have been carried out to explore the hypothesis that carbon monoxide exposure contributes to pathological changes leading to atherosclerosis(30). Pogeons(31), rabbits(32-34), dogs(34), primates(34-37), and man(38, 39) have all been investigated. No indications of any detrimental effects have so far been found at blood COHb levels of less than 15 %. The suggestion that carbon monoxide is implicated in the very weak statistical association between smoking and heart disease is very tenuous at best.

The recommendation by WHO of 4 % COHb as the maximum safe level must be considered as cautiously conservative. The evidence to date indicates that only if the individual is handicapped with severe heart disease might he have some difficulties at the 4 % COHb level (40, 41), but even then the probability is very low. Such a person may lack sufficient reserve to compensate for such loss in oxygen carrying capacity of the blood. Hence, if he were also exercising for a considerable period of time he might experience early onset of chest pains. (It is hardly likely that a room where people are smoking heavily is also a place where one would be engaging in strenuous exercise).

STEWARD (42) in his overall review of the carbon monoxide and smoking situation has published the information in Table 4 summarizing human responses to various levels of COHb (Table 4).

TABLE 4

Human Response to Elevated Carboxyhemoglobin in the Blood (42)

% COHb	Healthy Adult	Heart Patients
0.3 - 0.7	Normal	Normal
1 - 5	Increased blood flow	May feel lack of oxygen
2 - 9	Exercise tolerance reduced	Chest pains with less exertion
16 - 20	Headache, lowered visual response	May be lethal for patients with severe cardiac problems
20 - 30	Throbbing headache, nausea, impaired manual dexterity	
30 - 40	Severe headache, nausea, and vomiting	
50 -	Coma, convulsions	
67 - 70	Lethal	

CONCLUSIONS

The following conclusion which appears in the Workshop Report entitled Environmental Tobacco Smoke Effects on the Non-Smoker published in the Scandinavian Journal of Repiratory Diseases (43), is supported by many investigators who have studied this problem(44, 45).

Concentrations that are present under realistic environmental conditions may reach about 10 ppm. It is true that higher concentrations have been reported, but they represent only transient values or levels reached under experimental conditions. If the exposure to 10 ppm were to prevail for 8 hours the resulting COHb concentration would be 1.9 %. This value is well below the suggested WHO maximum of 4 %. At these levels no adverse biological effects will occur in a population that can be expected to be present in such environments. It can thus be concluded that the carbon monoxide in environmental tobacco smoke does not represent a health hazard.

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DEBATE

KRUSZYNSKI. Dr. Harke has published a study on changes in skin temperatures for active and passive smokers in extreme room conditions. I have not seen any confirmation of these findings anywhere. Have you seen a confirmation of Harke's findings or made own experiments on that item?

WAKEHAM. I know of no investigation to confirm Harke's findings.